Walk, Roger A.
From: Takada, Kohji
Sent: Sunday, April 09, 2000 11:18 AM
To: Walk, Roger A.
Subject: Japanese article for ARTIST

Dear Roger,

Regarding a Japanese article for ARTIST, I selected the following two papers as candidates. I am selecting the first one, because it is published in 1999, and the 2nd one is in 1998 and may be a bit too 'detailed' for ARTIST, although the quality seems better than the 1st one. I would appreciate your comments.

Best regards,
Kohji

Prenatal administration of nicotine results in dopaminergic alterations in the neocortex.
Muneoka K, Nakatsu T, Fuji J, Ogawa T, Takigawa M
Neurotoxicol Teratol 1999 Sep-Oct;21(5):603-9
This study examined the effect of two high doses (3 or 6 mg/kg/day) of nicotine administrations via injections to pregnant rats on the dopaminergic, serotonergic, and noradrenergic systems in six brain regions in young adult male rats. The 3 mg/kg/day and 6 mg/kg/ day nicotine exposure resulted in significant decreases in dihydroxyphenylacetic acid (DOPAC) content in the neocortex and in both the neocortex and in the midbrain plus pons medulla, respectively, without any effects on the other brain regions such as the hypothalamus or striatum. No significant effects of prenatal nicotine were found on norepinephrine, serotonin, or 5-hydroxy-3-indolacetic acid levels. These data demonstrated that prenatal nicotine induced disturbances in the dopaminergic system in the young adult period. Furthermore, the region-specific reductions in the DOPAC content suggests that the exposure to a high dose of nicotine in utero might cause a predisposition to diseases related to a dopaminergic dysfunction in the frontal cortex.

Nicotine administration decreases the number of binding sites and mRNA of M1 and M2 muscarinic receptors in specific brain regions of rat neonates.
Zhu J, Taniguchi T, Konishi Y, Mayumi M, Muramatsu I
Life Sci 1998;62(12):1089-98
Nicotine has been shown to delay the developmental increase of muscarinic receptors in brain of rat neonates. In this study, we have examined the muscarinic receptor binding sites and corresponding messenger RNA in rat neonates, whose mothers received nicotine in the drinking water, using competition binding study and northern blot analysis. On postnatal 14th day, nicotine treatment led to a 61% reduction in the proportion of high-affinity sites for pirenzepine (M1-subtype) to total [3H]quinuclidinyl benzilate binding sites in cerebral cortex in rat neonate. Nicotine treatment also decreased that for 11-2[[2-[(diethylamino)methyl]-1-piperidinyl]-5,11-dihydro-6H-pyrido [2,3-b] [1,4] benzodiazepin-6-one (AF-DX 116) (M2-subtype) by 77% in cerebellum on postnatal 14th day. The levels of m1 and m2 muscarinic receptor messenger RNAs in the same brain regions were also decreased in the same day after nicotine treatment. On postnatal 35th day, no difference was observed in binding sites or in messenger RNA levels of the brain regions tested (cerebral cortex, midbrain, hippocampus, cerebellum and brainstem). Our results show that nicotine reduces muscarinic receptor subtypes in developing rat brain, in part, through suppression of the messenger RNA expression, but that the effects are different among brain regions and subtypes.

Source: https://www.industrydocuments.ucsf.edu/docs/qhgj0001